Please amend the application as follows:

## In the Specification

Please replace the paragraph at page 1, line 13 through page 2, line 16, with the following paragraph:

Lyme disease begins at the site of a tick bite, producing a primary infection with spread of the organism to secondary sites occurring early in the course of infection. Lyme disease is a progressive multi-system disorder and is the most common vector-borne disease in both North America and Europe. This disease was first described as a focus of pediatric arthritis patients in Old Lyme, CT (Steere, A.C., et al., Arth. Rheum., 20:17 (1977)). The association of this syndrome with the bite of the deer tick, Ixodes scapularis, led to the identification of the spirochete Borrelia burgdorferi as the causative agent (Burgdorfer, W., et al., Science, 216:1317-1319 (1982)). As culture isolation of the bacterium from clinical and field samples became more efficient, Baranton and colleagues described three pathogenic genospecies, B. burgdorferi sensu stricto (B. burgdorferi or B.b.s.s.), B. afzelii, and B. garinii (Baraton, G., et al., Int. J. Syst. Bacteriol., 42:378-383 (1992)). These are members of a species complex, B. burgdorferi sensu lato, which consists of at least 10 different genospecies (Piken, R.N., et al., J. Invest: Dermatol., 110:211-214 (1998); Postic, D., et al., Int. J. Syst. Bacteriol., 44:743-752 (1994); Valsangiacomo, C.T., et al., Int. J. Syst. Bacteriol., 47:1-10 (1997)). B. burgdorferi, B. afzelii and B. garinii are thought to be pathogenic and all are found in Europe, but in North America, B. burgdorferi is the only pathogenic genospecies found. Each of these three genospecies is associated with distinct clinical manifestations (Van Dam, A. P. et al., Clin. Infect. Dis., 17:708-717 (1993)). This implies that differences in genospecies may play an important role in the wide array of clinical manifestations observed in Lyme Disease.



Please replace the paragraph at page 10, lines 18-26, with the following paragraph:

There is evidence that ospC has been transferred between strains and even between genospecies (Wang I-N, et al., Genetics, 151:15-30 (1998)). This is not true of Borrelia chromosomal genes (Dykhuizen, D.E., et al., Proc. Natl. Acad. Sci., 30:10163-10167 (1999); Maynard Smith, J. and Smith, N.H., Mol. Biol. Evol., 15:590-599 (1998)). However, ospA and ospC alleles in B. burgdorferi sensu stricto are almost completely linked (Wang I-N, et al., Genetics, 151:15-30 (1999)). This suggests that once an ospC allele has been transferred into a particular background, there is little or no selection for another similar recombination event. Thus, each major ospC group represents a clonal population descended from a single recombination.

Please replace the paragraph at page 43, lines 1-25, with the following paragraph:

TABLE VII

OspC Polypeptides and Chimeric Polypeptides of the Present Invention

YPEPTIDE SEQ ID NO.:(DNA) (POLYPEPTIDES)

POLYPEPTIDE	SEQ ID NO.:(DNA)	(FOLTET TESES)
	45	46
unlip OspC kkp(55-621*)	47	48
unlip OspC PKO		50
unlip OspC TRO	49	
²unlip OspC-55B31/	51	52
58PKO/56TRO		54
unlip OspC1-TRO	53	
unlip OspC-TRO	55	56
	57	58
<sup>3</sup> Blip OspC1C10		60
BlipOspC12	59	76
Blip OspC1-TR0	75	
	65	66
Blip OspC2C7	. 61	62
Blip OspC2C10		_

